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PHYSIOLOGY

QUESTION 1

DISCUSS THE LONG TERM REGULATION OF MEAN ARTERIAL BLOOD PRESSURE

The long-term level of arterial pressure is dependent on the relationship between arterial pressure and the urinary output of salt and water, which, in turn, is affected by a number of factors, including renal sympathetic nerve activity (RSNA). In the present brief review, we consider the mechanisms within the brain that can influence RSNA, focusing particularly on hypothalamic mechanisms. The paraventricular nucleus (PVN) in the hypothalamus has major direct and indirect connections with the sympathetic outflow and there is now considerable evidence that tonic activation of the PVN sympathetic pathway contributes to the sustained increased level of RSNA that occurs in conditions such as heart failure and neurogenic hypertension. The tonic activity of PVN sympathetic neurons, in turn, depends upon the balance of excitatory and inhibitory inputs. A number of neurotransmitters and neuromodulators are involved in these tonic excitatory and inhibitory effects, including glutamate, GABA, angiotensin II and nitric oxide. The dorsomedial hypothalamic nucleus (DMH) also exerts a powerful influence over sympathetic activity, including RSNA, via synapses with sympathetic nuclei in the medulla and, possibly, also other brainstem regions. The DMH sympathetic pathway is an important component of the phasic sympathoexcitatory responses associated with acute stress, but there is no evidence that it is an important component of the central pathways that produce long-term changes in arterial pressure. Nevertheless, it is possible that repeated episodic activation of this pathway could lead to vascular hypertrophy and, thus, sustained changes in vascular resistance and arterial pressure. Recent studies have reactivated the old debate concerning the possible role of the baroreceptor reflex in the long-term regulation of sympathetic activity. Therefore, central resetting of the baroreceptor-sympathetic reflex may be an important component of the mechanisms causing sustained changes in RSNA. However, little is known about the cellular mechanisms that could cause such resetting.

QUESTION 2

WRITE SHORT NOTES ON THE FOLLOWING

- 1. PULMONARY CIRCULATION**
- 2. CIRCLE OF WILLIS**
- 3. SPLANCHNIC CIRCULATION**
- 4. CORONARY CIRCULATION**
- 5. CUTANEOUS CIRCULATION**

PULMONARY CIRCULATION

Pulmonary circulation, system of blood vessels that forms a closed circuit between the heart and the lungs, as distinguished from the systemic circulation between the heart and all other body tissues. On the evolutionary cycle, pulmonary circulation first occurs in lungfishes and amphibians, the first animals to acquire a three-chambered heart. The pulmonary circulation becomes totally separate in crocodilians, birds, and mammals, when the ventricle is divided into two chambers, producing a four-chambered heart. In these forms the pulmonary circuit begins with the right ventricle, which pumps deoxygenated blood through the pulmonary artery. This artery divides above the heart into two branches, to the right and left lungs, where the arteries further subdivide into smaller and smaller branches until the capillaries in the pulmonary air sacs (alveoli) are reached. In the capillaries the blood takes up oxygen from the air breathed into the air sacs and releases carbon dioxide. It then flows into larger and larger vessels until the pulmonary veins (usually four in number, each serving a whole lobe of the lung) are reached. The pulmonary veins open into the left atrium of the heart.

CIRCLE OF WILLIS

The circle of Willis is an important junction of arteries at the base of the brain. The structure encircles the middle area of the brain, including the stalk of the pituitary gland and other important structures. Two arteries, called the carotid arteries, supply blood to the brain. They run along either side of the neck and lead directly to the circle of Willis. Each carotid artery branches into an internal and external carotid artery. The internal carotid artery then branches into the cerebral arteries. This structure allows all of the blood from the two internal carotid arteries to pass through the circle of Willis.

The structure of the circle of Willis includes:

- left and right internal carotid arteries
- left and right anterior cerebral arteries
- left and right posterior cerebral arteries
- left and right posterior communicating arteries
- basilar artery
- anterior communicating artery

The circle of Willis is critical, as it is the meeting point of many important arteries supplying blood to the brain. The internal carotid arteries branch off from here into smaller arteries, which deliver much of the brain's blood supply.

SPLANCHNIC CIRCULATION

The splanchnic circulation is composed of gastric, small intestinal, colonic, pancreatic, hepatic, and splenic circulations, arranged in parallel with one another. The three major arteries that supply the splanchnic organs, celiac and superior and inferior mesenteric, give rise to smaller arteries that anastomose extensively. The circulation of some splanchnic organs is complicated by the existence of an intramural circulation. Redistribution of total blood flow between intramural vascular circuits may be as important as total blood flow. Numerous extrinsic and intrinsic factors influence the splanchnic circulation. Extrinsic factors include general hemodynamic conditions of the cardiovascular system, autonomic nervous system, and circulating neurohumoral agents. Intrinsic mechanisms include special properties of the vasculature, local metabolites, intrinsic nerves, paracrine substances, and local hormones. The existence of a multiplicity of regulatory mechanisms provides overlapping controls and restricts radical changes in tissue perfusion.

CORONARY CIRCULATION

Coronary circulation is the circulation of blood in the blood vessels that supply the heart muscle (myocardium). Coronary arteries supply oxygenated blood to the heart muscle, and cardiac veins drain away the blood once it has been deoxygenated. Because the rest of the body, and most especially the brain, needs a steady supply of oxygenated blood that is free of all but the slightest interruptions, the heart is required to function continuously. Therefore its circulation is of major importance not only to its own tissues but to the entire body and even the level of consciousness of the brain from moment to moment. Interruptions of coronary circulation quickly cause heart attacks (myocardial infarctions), in which the heart muscle is damaged by oxygen starvation. Such interruptions are usually caused by

ischemic heart disease (coronary artery disease) and sometimes by embolism from other causes like obstruction in blood flow through vessels.

CUTANEOUS CIRCULATION

Cutaneous blood flow is regulated mainly by body temperature. Hypothalamus plays an important role in regulating cutaneous blood flow. When body temperature increases, the hypothalamus is activated. Hypothalamus in turn causes cutaneous vasodilatation by acting through medullary vasomotor center. Now, blood flow increases in skin. Increase in cutaneous blood flow causes the loss of heat from the body through sweat. When body temperature is low, vasoconstriction occurs in the skin. Therefore, the blood flow to skin decreases and prevents the heat loss from skin.

FUNCTIONS

Supply of nutrition to skin

Regulation of body temperature by heat loss

QUESTION 3

DISCUSS THE CARDIOVASCULAR ADJUSTMENT THAT OCCURS DURING EXERCISE

The integrated response to severe exercise involves fourfold to fivefold increases in cardiac output, which are due primarily to increases in cardiac rate and to a lesser extent to augmentation of stroke volume. The increase in stroke volume is partly due to an increase in end-diastolic cardiac size (Frank-Starling mechanism) and secondarily due to a reduction in end-systolic cardiac size. The full role of the Frank-Starling mechanism is masked by the concomitant tachycardia. The reduction in end-systolic dimensions can be related to increased contractility, mediated by beta adrenergic stimulation. Beta adrenergic blockade prevents the inotropic response, the decrease in end-systolic dimensions, and approximately 50% of the tachycardia of exercise.

The enhanced cardiac output is distributed preferentially to the exercising muscles including the heart. Blood flow to the heart increases four-fold to fivefold as well, mainly reflecting the augmented metabolic requirements of the myocardium due to near maximal increases in cardiac rate and contractility. Blood flow to the inactive

viscera (e.g., kidney and gastrointestinal tract) is maintained during severe exercise in the normal dog. It is suggested that local autoregulatory mechanisms are responsible for maintained visceral flow in the face of neural and hormonal autonomic drive, which acts to constrict renal and mesenteric vessels and to reduce blood flow. However, in the presence of circulatory impairment, where oxygen delivery to the exercising muscles is impaired as occurs to complete heart block where normal heart rate increases during exercise are prevented, or in congestive right heart failure, where normal stroke volume increases during exercise are impaired, or in the presence of severe anemia, where oxygen-carrying capacity of the blood is limited, visceral blood flows are reduced drastically and blood is diverted to the exercising musculature. Thus, visceral flow is normally maintained during severe exercise as long as all other compensatory mechanisms remain intact. However, when any other compensatory mechanism is disrupted (even the elimination of splenic reserve in the dog), reduction and diversion of visceral flow occur.